April 4, 1950.

Dr. D. M. Bonner,
Yale OBL
New Haven, Conn.

Dear Dave:

I am very glad to hear what progress you are making- the beneficent stimulation by Stadler could certainly have been predicted. Give him our best.

It is certainly welcome to hear about some real genetic work being done on Neurospora. Has enough groundwork been laid so that you know what linked factors to use to check the correlation of the nic$^-$ mutation with crossingover? I've had a somewhat analogous situation in coli: Lac_1^- and Lac_4^- crossover considerably less than 0.1%. However, they yield a Lac$^+$ diploid heterozygote. In general, they can also be distinguished by their action on butyl galactoside. But at first sight, they were regarded as allelic. There is one point on heterokaryons that I have been a little fanatical about: were your tests negative because of the ineffectiveness of a heterokaryon, or was no heterokaryon formed? I have always felt that independent evidence (viz. other markers) should be brought up for actual heterokaryon formation whenever this is critical.

There is one other problem that may now be possible to attack in Neurospora. A great many other fungi show evidence, more or less secure, for delayed reduction, occasionally resulting in diploid mycelia. Using closely linked factors, which should interact to give the wild type in a double heterozygote, one should be able to select more or less effectively for such exceptions. Comparison of heterokaryons would really be an interesting problem. with heterozygotes.
I bring this up only as an analogy to the coli diploids.

If I am not mistaken, Stadler is pretty much of the same mind as we are on gene-enzyme relationships. Would it be too unwieldy for all three of us to join forces?

Sincerely,

Joshua Lederberg